treating carotid disease, medically or surgically, is to prevent stroke—the rare exceptions being where global ischemia causes disabling symptoms. Studies that focus on late survival as an end point and that do not randomly assign symptomatic patients according to the degree of carotid stenosis or ulceration are numerous. Such studies beg the question of whether there is a subset of patients who have experienced transient ischemic attacks or a limited completed stroke and who have a territorially appropriate carotid lesion and can have their chance of future stroke diminished by a particular form of therapy.

The medical treatment of cerebrovascular disease involves the control of hypertension and antiplatelet therapy. Hypertension remains a critical factor in cerebrovascular morbidity. It has been suggested that improved hypertension treatment is responsible for the decrease in the national stroke rate since 1970, but this remains unproved. Antiplatelet therapy has become a mainstay of the treatment of transient ischemic attacks despite most studies showing no stroke-preventive benefit over placebo, only decreased late mortality; 15% to 20% of patients in the "aspirin trials" have gone on to completed stroke within two years.

Scattered reports have appeared of intraluminal balloon angioplasty of carotid bifurcation atherosclerotic stenoses and internal carotid fibromuscular disease. Close monitoring and careful evaluation will be necessary before welcoming or condemning this technique.

Operative intervention for symptomatic carotid disease has been shown by numerous studies to substantially benefit patients with significant stenotic or ulcerative lesions and ipsilateral ischemic events. The stroke rate following a carotid operation would appear to be 1.0% to 1.8% per year for patients with hemispheric symptoms and lower for patients presenting with amaurosis fugax. The comparative stroke rate for patients with transient ischemia treated medically is at least 5% a year. It should be recalled that this figure includes patients with no or minimal demonstrable carotid disease and almost certainly underestimates the natural history of transient ischemic attacks in that cohort with significant carotid disease. Published perioperative stroke and mortality statistics indicate that a carotid operation can be done safely—combined stroke and death rate less than 4%—by experienced surgeons with the proper patient selection. Some series have shown unacceptable results. It is incumbent on each surgeon who does carotid surgery to approach this procedure only with the appropriate indications, to operate with the utmost respect for the potential morbidity, and to constantly review his or her own statistics.

Carotid bifurcation endarterectomy in well-selected cases will result in the relief of hemispheric symptoms with near certainty, a lower recurrent stroke rate for those patients with limited fixed deficits, and the relief of basivertebral and global symptoms in 50% to 70% of cases. The best results in this last category are obtained in patients with classic posterior circulation symptoms, a highly stenotic internal carotid artery, and angiographic posterior circulation compromise. External carotid reconstruction may help selected patients with internal carotid occlusion and definite hemispheric symptoms, particularly if the middle cerebral artery is supplied from that stenotic artery angiographically. Patients with hemispheric transient ischemic attacks and relatively normal carotid arteriograms appear to be at a lower risk for late stroke, particularly if they have had only a single event. Those with multiple events require more careful follow-up with noninvasive examinations.

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Bleeding, Clotting, and the Use of Blood Products in Trauma Care

IMMEDIATELY AFTER SERIOUS INJURY, the ability to limit bleeding from injured tissues largely determines the outcome. For trauma surgeons, there are few situations more harrowing than dealing with a seriously injured patient who has had a breakdown in hemostatic mechanisms. Various approaches designed to prevent such a loss of hemostatic control have been proposed, and some have gained considerable popularity. Whether they are scientifically correct and clinically beneficial is sometimes doubtful.

Several factors are known or suspected to contribute to a breakdown in hemostasis in an injured patient. Preexisting major abnormalities are fortunately rare and usually related to the use of therapeutic drugs. Anticoagulation with vitamin K antagonists is one of the few legitimate indications for the prophylactic use of fresh frozen plasma. Significant hepatocellular insufficiency is another. Hypothermia is one of the avoidable causes of impaired hemostasis and is probably one of the most underestimated in its importance. Hypothermia impairs various steps in the coagulation sequence and seems to have a remarkably strong effect on platelet function in vivo. Hypoperfusion is emerging as perhaps the key causal element in those few patients who have a true systemic breakdown in hemostasis. This concept has been strongly confirmed by almost all the experimental work in this area and has recently been supported by clinical data. The sooner bleeding is stopped and the blood volume restored, the less probability there is of a coagulopathy developing. This effect of sustained and severe hypoperfusion accounts, at least in part, for the association of impaired hemostasis with a massive transfusion.

Massive transfusion deserves special comment because it has become the focus of prophylactic interventions. Stored blood is variably deficient in some coagulation factors (primarily V and VIII) and is totally devoid of functioning platelets. Therefore, a dilutional or washout effect is anticipated and much feared. Various detailed studies of heavily transfused patients have not shown clinically significant coagulation component deficits, however, except in those with clinically obvious impaired hemostasis in whom the deficits are much greater than can be accounted for by dilution. Factors V and VIII are particularly well maintained because of their rapid rates of synthesis, but these are the two that are depleted in stored blood. Platelets are of a more rational concern, and the platelet count is certainly driven down by dilution in heavily transfused patients. Almost all studies in transfused patients and the pertinent studies in patients un-

dergoing therapeutic plasmapheresis show great difficulty in driving the platelet count below 50×10^9 per liter (50,000 per μl) in patients with intact marrow function. Most important, the hypothesis that giving platelets prophylactically will make a difference in seriously injured, heavily transfused patients has been tested in a randomized prospective trial in Seattle. There was little effect of the prophylactic administration of platelets on the platelet count and no effect on any criterion for bleeding.

The main reason to be selective is that the use of fresh frozen plasma and platelets can transmit serious or lethal infections. The acquired immunodeficiency syndrome has the public's attention at the moment. More important is transfusion-transmitted non-A, non-B hepatitis. It is highly doubtful that the prophylactic administration of fresh frozen plasma and platelets in most transfused injured patients does more good than harm, an opinion recently affirmed by the consensus conferences of the National Institutes of Health.

A recognition of the risk of disease transmission with transfusion has altered the practice of most trauma surgeons. There is now greater emphasis on blood salvaging techniques and autotransfusion. There is general acceptance that lower hematocrits are safe in the average young trauma patient, and the old rule of giving "2 units" of blood automatically when a transfusion is required has been—or should be—abandoned. Many surgeons now question the wisdom of the nonoperative management of solid organ injuries, as patients with these injuries average twice the number of transfusions as those treated surgically.

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Surgical Treatment of Peptic Ulcer Disease

ALTHOUGH THE FREQUENCY of hospital admissions for peptic ulcer disease was decreasing even before the introduction of H_2-receptor antagonists in the United States in 1977, since 1977 the number of operations for peptic ulcer disease has decreased considerably. In addition to H_2-receptor antagonists, sucralfate (a surface-active agent) has been increasingly used in recent years for the medical management of peptic ulcer disease. In the near future, proton pump blockers will provide nearly complete control of acid secretion. In the face of improved medical management, the nature of the surgical treatment of peptic ulcer disease has changed. Overall, fewer operations are being done than in previous years. A greater percentage of patients are being operated on for the complications of peptic ulcer disease—hemorrhage, perforation, and pyloric obstruction—than for intractable peptic ulcer disease. Those patients who are treated surgically tend to be older and sicker than in previous years, with resultant increased morbidity and mortality.

In addition to the classic operations for peptic ulcer disease—antrectomy with truncal vagotomy and truncal vagotomy with a drainage procedure—the proximal gastric vagotomy (PGV) has been increasingly used for peptic ulcer disease over the past few decades. Although its recurrence rate approaches 15%, the PGV offers less postoperative morbidity (diarrhea and dumping) than the classic operations. For this reason, many consider it the operation of choice for intractable, uncomplicated duodenal ulcer disease. The PGV is also being increasingly used in patients with perforated duodenal ulcers who are considered candidates for a definitive ulcer operation at the time of plication. It has been recommended by some investigators for use in bleeding or obstructing duodenal ulcers and in gastric ulcers but still must be viewed as unproved in these cases. For bleeding duodenal ulcers, oversewing of the bleeding vessel with the truncal vagotomy and pyloroplasty or truncal vagotomy and antrectomy remains the standard. For obstructing duodenal ulcer, a truncal vagotomy and gastrojejunostomy or truncal vagotomy and antrectomy remain the standard operations. For type I gastric ulcers—those to the left of the gastric angulus without associated pyloric or duodenal disease—antrectomy without vagotomy remains the standard operation. For prepyloric ulcers, the PGV has a high recurrence rate, and most surgeons would favor the use of a truncal vagotomy and antrectomy.

Although surgical practice in peptic ulcer disease has changed in recent years, general surgeons must be familiar with all of the operations for peptic ulcer disease and tailor the operation to the given situation of each patient.

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Vascular Endoscopy

ENDOSCOPY OF THE VASCULAR SYSTEM has evolved over recent years from an experimental research procedure to a sophisticated diagnostic and therapeutic adjunct for surgical or percutaneous interventions of the peripheral vascular and coronary circulation. Advances in fiber optics and medical electronic imaging systems now allow the production of flexible angioscopy catheters, which incorporate a fluid irrigation channel, in diameters of 2 mm. Ultrafine instruments down to 0.2 mm are also being investigated.

Intraoperative angioscopy is done by introducing the endoscopic catheter through the vessel incision made for a bypass or other procedures. An objective lens transmits the image through a coherent bundle of several thousand quartz fibers to a video camera and magnification system, with a color image projected onto a TV monitor. Flushing of a heparinized saline solution through the irrigation channel clears blood from the field of view and provides a superb three-dimensional view of the vessel interior.

Applications include the verification or further elucidation of ambiguous angiogram abnormalities—including more accurately estimating the severity of stenosis and plaque morphology and differentiating thrombotic occlusion from atherosclerotic processes—the inspection of anastomotic suture lines to exclude technical faults, the identifica-